fic in character; sometimes there is an alteration of protoplasm (chromatolysis, vacuolization, and atrophy) or of the nucleus (atrophy, disappearance); sometimes an inflammation of the pericellular capsule with hyperplasia of the cells or of the connective tissue. The following conclusions are noted as the results of this very interesting study:

The essential lesion of tabes is after all a dystrophy, which occurs in the whole of the peripheral sensory neurone, predominating especially in the central prolongation of the cell. This dystrophy attacks generally certain portions of the motor protoneurone and of the sympathetic system. The histological alterations are characterized by modifications in the nerve fibers which can be compared under certain conditions to those which occur in the course of a toxic neuritis, either produced experimentally or in the course of disease. They are very distinct from a Wallerian degeneration. If the cell body of the sensitive protoneurone appears in general to keep its structure and its normal aspect, it is nevertheless sometimes the seat of a trophic lesion. In addition, in spite of the absence of atomic lesions, it is reasonable to suppose that its trophic function is in a certain measure compromised.

4. *Bromide Action.*—This paper is a continuation of the experiments made by Féré to determine the effects of doses of bromide upon the work capacity. The cortex of the brain gives rise to voluntary muscular activity upon excitation, and the ergograph of Mosso is the means used to measure this. The mass of experimental data makes an abstract of this article an impossibility.

5. *Does a Peroneal Form of Charcot Marie Amyotrophy Exist?*—This question is answered in the affirmative by Sainton in a brief communication in which he mentions two cases of amyotrophy limited to the lower limbs. The grandmother of these individuals had the same condition which was limited as in them to the lower extremities. Although there has been no autopsy on such cases, the existence of a peroneal amyotrophy of the Charcot-Marie type cannot be doubted.

SIDNEY I. SCHWAB (St. Louis).

## AMERICAN JOURNAL OF INSANITY.

(Vol. LVIII, 1902, No. 4.)

1. Etiology of Paresis. HURD.
2. The Early Diagnosis of Paresis. F. X. DERCUM.
3. The Comparative Frequency of General Paresis. O. G. WAGNER.
4. Treatment of Paresis; Its Limitations and Expectations. E. COWLES.
5. Heredity—With a Study of the Statistics of the New York State Hospitals. W. G. KRAUSS.
6. Senility and Senile Dementia. RUSSELL.
7. Some Observations upon the Elimination of Indican, Acetone and Diacetic Acid in Various Psychoses. CORIAT.
8. Studies in the Manic-Depressive Insanity, with Report of Autopsies in Two Cases. S. PATON.

1. *Etiology of Paresis.*—The author takes up the various factors bearing on the etiology of this disease, beginning with syphilis, citing the results of observations of different writers, which vary all the way from 11 to 94 per cent., while in his own cases he has found only 20 per cent. of paretics syphilitic, which he accounts for largely by the inability to secure a complete anamnesis in many cases of the disease. As a further argument in favor of lues, he shows that this disease is particularly rare where syphilis does not exist, as in Ireland; but again in Egypt and Japan, where it is extremely prevalent, these countries lack the special stress coincident with Western civilization. Further clergy, priests and Quakers, who are rarely syphilitic, are proportionately free from paresis. It has also been observed,

that paresis is much less prevalent in rural districts, where lues and mental stress are rare. He calls attention to the like relation of syphilis and paresis in men and women, *i.e.,* about 4 to 1. In juvenile cases hereditary syphilis has been found to the extent of 100 per cent. He ascribes little weight to heredity and considers paresis one of the least transmissible of mental diseases. He regards alcohol more as an incidental factor, and inebriety is often developed after the onset of the disease and therefore more a symptom than a cause. With respect to head injuries, they have no special etiological bearing and the effects of lead, tobacco, *etc.,* he regards as problematical. He believes the increased mental anxiety and over-exertion of the century just past is largely responsible for the startling increase in the frequency of paresis. The similarity of tabes and paresis has been urged in favor of the syphilitic etiology of paresis, for in the former disease a history of luetic infection is found in 80 to 90 per cent. of the cases. The failure of anti-specific treatment in paresis is ascribed to the different tissues affected in brain syphilis and paresis, in the latter the neurone, in the former the meninges, and the latter is incapable of regeneration. He concludes his paper: "That syphilis is the most common factor in the production of paresis. That it may cause it directly—an exciting cause. That it may cause it indirectly by bringing about such a devitalization of the system generally as to render other influences operative—a predisposing cause. That it is not usually the sole cause, but that there is associated with it the deleterious effect of mental stress and over-excitement, dissipation and alcoholism, and heredity. That in a certain relatively small number of cases mental stress, worry or overwork may be the sole ascertainable cause. That traumatism may also be the cause in a still smaller proportion of cases, but that in many of them it acts as a developing or ripening agent of an incipient paresis in a syphilitic subject."

2. *The Early Diagnosis of Paresis.*—Differentiation from neurasthenia difficult in the early stage, yet the symptoms only superficially similar, those of the neurasthenic being more subjective, while the paretic rarely complains or consults a physician, his friends narrate his symptoms. In neurasthenia the physical appearance rarely changed, or if so, differently from paresis, where the facial expression is changed in quality. The neurasthenic's movements are not altered in precision, not incoördinate. The neurasthenic's sleep disorders nearly the opposite of the paretic's; the neurasthenic is at his best in the evening, the opposite with the paretic, for then his little delinquencies in the matters of dress, deportment and speech defects become more prominent. Quantitative mental changes occur early in paresis, not so in neurasthenia; the neurasthenic is distinctly nosophobic, has no essential memory defect, still mental functions quickly tire. In paresis early mental fatigue, but loss of ability to remember and to comprehend is added; soon marked impairment of memory, manifested by neglect of attire, business, various improprieties, *etc.,* which are foreign to neurasthenia. The loose conception of neurasthenia largely chargeable for any confusion with paresis. This early stage he terms the neurasthenoid. Paresis may be confused with hypochondria and melancholia. Same factors distinguish it from hypochondria as from neurasthenia. Physical symptoms to be early looked for: Visual field may show some contraction at a very early period, but there is no retinal hyperesthesia, as in neurasthenia. Sequence of physical signs immaterial. The "paretic manner" so hard to describe, precedes all other physical signs, consisting of slight changes in facial expression and gesticulation: "that something in the eye, the face, the gestures, the words, which reveal that the patient is not in close and accurate touch with his surroundings, with his business affairs, with the fact of his illness, or for that matter, with any subject." Significance of specific history obvious. Very difficult to differentiate from alcoholism, for alcoholic excess is one of the

early symptoms of paresis. In alcoholism obtusion to proprieties and moralities soon cease after discontinuance of the alcohol. If alcoholic dementia exists, it is of the confusional type. General appearance of alcoholism indicative. In brain syphilis symptoms of organic lesion, diffuse, or limited, without quantitative mental changes. Early differentiation between tabes and paresis may be impossible, for many cases beginning apparently as tabes end in paresis. Absence of mental symptoms distinctive. Careful observation will generally lead to diagnosis in neurasthenoid stage.

3. *Comparative Frequency of General Paresis.*—Paresis, a disease known only about eighty years, being first recognized in this country at McLean Asylum by Dr. Bell in 1843. Statistics collected from various sources since 1849 show there has been a gradual increase from 1.5 to 12 per cent. at present of all admissions, and is everywhere from 5 to 8 times more frequent in men than in women, varying according to the urban or suburban character of the patients in the institution. The London Asylum reports show a percentage of 12 and 6 men to one woman affected, as well as that more married than unmarried people have the disease. It affects all classes and representatives of all vocations are found among its victims. Data from the Manhattan State Hospital East show a total average duration of a little more than two years and that native Americans are more prone to the disease than foreigners. It is further shown that the disease is rare before twenty and after sixty. From all sources of information it is found that this disease affects about 8.75 per cent. of all insane, is most frequent between thirty and fifty, is gradually increasing in frequency, men seven times more liable to the disease than women, is invariably fatal, and lasts less than two and a half years. Is nearly twice as frequent in a city population as in that of the rural districts. No special class subject to the disease, but wherever mental stress is combined with more or less hereditary influence. Overwork, sexual excesses, alcoholism, irregular mode of life, sunstroke, and cerebral traumatism seem to be special factors in its production.

4. *Treatment of Paresis; Its Limitations and Expectations.*—Neurological, histological and pathological findings present a barren field of limitations with respect to therapeutics. Pathological research with the microscope has nearly reached its limit, while physiological chemistry may be able to solve many clinical problems as yet beyond our ken. Metabolic changes must precede visible structural changes is a self-evident fact, and these metabolic changes are chemical within the molecules. From the etiological standpoint we are again limited therapeutically, as antisyphilitic treatment has generally proven futile. Heredity is of minor importance as an etiological factor. Alcohol may be a contributing cause by lowering the normal resistance to morbid influences, and therefore etiology affords no guide to treatment. The expectations of treatment offer an interesting field. While the relation between syphilis and paresis is unknown, it is probable there is some intervening factor or condition uniting them, for without syphilis, mental stress, excitement and excesses lead to neurasthenia, while the syphilitic neurasthenic generally becomes a paretic. A state of toxemia seems in all probability to represent this intervening condition. Drs. Mott, Kraepelin and Wernicke recognize the toxic agency in the etiology of paresis, which has induced a degeneration of the neurone, and this toxic agency may be of bacterial origin. It is a well established fact that auto-intoxications disturb the intimate harmony of brain cell metabolism, but this field is still one largely unexplored. Dr. Bruce and Dr. Ford Robertson have recently called attention to the exacerbations of this disease being accompanied by gastric and intestinal disorders, rise of temperature and hyperleucocytosis. By injecting serum obtained from a paretic during a remission Dr. Bruce claims to have effected a complete remission in two cases. Dr.

Bruce's conclusions are: "(1) General paresis is a disease directly due to poisoning by the toxins of bacteria, whose point of attack is through the gastric and intestinal mucous membrane. (2) The poisoning is probably a mixed poisoning, but the bacillus coli is apparently one of the noxious organisms. (3) The result of treatment with a serum taken from a case of general paresis in a condition of remission and injected subcutaneously into an early progressive case, points strongly to the fact that some form of serum treatment is the proper one." Dr. Robertson believes this disease depends on the occurrence of chronic toxemia of gastro-intestinal origin, due to a failure of the natural alimentary bacteria, thus giving rise to toxins whose absorption leads to vascular changes in the brain and so degeneration of the nerve elements; syphilis merely altering the natural immunity, and it is along this line Dr. Cowles believes that future researches may lead to alleviation of this disease.

5. *Heredity*—With a Study of the Statistics of the New York State Hospitals.—After defining the various forms of heredity and giving the statistics bearing on heredity from the several State Hospitals of New York, he summarizes by showing heredity in 25.8 to 39.7 per cent. of all cases. Nothing new is offered as to nervous diseases and the article concludes with a classification of nervous disease under direct, indirect heredity and those with no heredity.

6. *Senility and Senile Dementia.*—Senile cases constitute about 23 per cent. of the admissions to the Willard State Hospital. The writer recites the signs of senile decay, both mental and physical, in that there is a general blunting of the mental functions and a deterioration of the physical processes, both of which may remain within normal limits. There are wide variations as to the age when these changes appear and in the rapidity of their progression, yet sixty is regarded about the average. Temperament and diathesis are the prime factors which may cause senility to become of a pathological nature, as they largely govern the individual's resistance to pernicious factors, like the infections and toxins, and they thus induce senile dementia. A careful examination will generally be competent to separate normal senility from senile dementia, and of its symptoms the most important, aside from hallucinations and fixed delusions, are slight exaltation and restlessness, degraded, filthy habits, erotic tendencies, destructiveness and marked perversity in conduct. Insomnia may also be a prominent symptom.

7. *The Elimination of Indican, Acetone and Diacetic Acid in Various Psychoses.*—Indican seems to be increased in depressive states, particularly when attended by auto-intoxication, and its elimination decreases when the latter condition is overcome. The author gives a ready method for determining the relative amount of indican. The deductions of different writers are summarized as to the significance of indican, acetone and diacetic acid and their excess in the urine is generally accepted to be an indication of defective metabolism and coincident with auto-intoxication. Excess of acetone is said to occur in states of fear, but this the author has been unable to confirm. The abstracts of 26 cases are given, all of which have depressive phases, and a summary shows that indican is found to be coincident with katatonic and epileptic stupor, akinetic forms of dementia præcox and general paresis, as well as in alcoholic depression and depressive phases of the manic-depressive psychosis, or any disease where akinesis is a prominent symptom. On the other hand there is a lessened elimination of indican in katatonic excitement, exhilaration of general paresis, as well as in exalted states of dementia præcox and in the manic phases of the manic-depressive psychosis, or in other words in states of hyperkinesis. These variations in indican elimination are proven to be wholly independent of stools, diet and weight. Acetone was found in all the cases except epileptic

stupor, diacetic acid in melancholia, the akinetic conditions of dementia præcox and general paresis, alcoholic hallucination with fear. There appears to be no definite relation between the production of these bodies and the psychoses.

8. *Studies in the Manic-Depressive Insanity.*—Careful and systematic study of these cases reveals the presence of certain symptoms which form the basis of a common clinical complex whose variations are much more quantitative than qualitative changes. Use of the terms "mania" and "melancholia" smacks of casuistry, and the careful study of these conditions discloses their intimate relationship, the manic states generally being preceded by depression. Several cases are cited to elucidate this fact and they show that manic and depressive conditions are not antithetical. Further special study should be made of such cases as to blood pressure and the changes in the urine during the periods of excitement and depression.

McCorn (New York).

### REVUE NEUROLOGIQUE.

(Vol. X, 1902, No. 11, June 15.)

1. Primary Bronchial Cancer, followed by Miliary Carcinosis with Polyneural Complications. J. Oberthur.

1. *Polyneural Complications in Bronchial Cancer.*—An account of what the author believed to be a case of primary carcinoma of the left lung, having a bronchial origin, which rapidly invaded the right lung. The epithelial elements entering the general circulation through the pulmonary vein, were thus distributed throughout the entire system, and the cancer of the lung was most highly favored by a corresponding infection of the entire organism. The rarity of such a development, the large number and great variety of the metastases, the absence of any large ganglion accessible to palpation, the preponderance of motor and sensory phenomena, the difficulty of examination, the unusual progress of the disease, all these factors easily explain why a satisfactory diagnosis was impossible during life. The lesions of the neuro-muscular system were of two kinds; beside the ordinary lesions of cancerous cachexia, lesions of a mechanical nature were also present, on the one hand considerable compression and destruction of the motor and sensory nerve endings, and on the other hand general cancerous muscular inflammation, which together gave a particularly painful character to the polyneural complications of this peculiar pathogenesis.

Jelliffe.

### BRAIN.

(Vol. 25, 1902, No. 98, Summer.)

1. On Pachymeningitis Hæmorrhagica Interna. J. O. W. Barratt.
2. Case of Tubercular Tumor of the Spinal Cord in a Child Two Years Old. W. K. Hunter.
3. A Contribution to the Pathology of Acute Insanity. David Orr.
4. Uniradicular Palsies of the Brachial Plexus. E. Farquhar Buzzard.
5. A Case of Sclerotic Atrophy of Cerebrum and Cerebellum, Familial Type, Occurring in a Boy. J. Mitchell Clarke.
6. The Pathology of So-Called Acute Myelitis. H. Douglas Singer.
7. A Case of Acromegaly under Observation for Five Years, with Charts of the Fields of Vision. F. Richardson Cross.

1. *Internal Hemorrhagic Pachymeningitis.*—The author summarizes his observations as follows: (1) Intravascular separation of fibrin is an apparently constant event in subdural membrane formation. (2) Subdural membranes are free from micro-organisms as revealed by staining methods. The results of attempted culture upon agar, though not always negative, indicate that during life such membranes are free from bacteria. (3) Experimental implantation of false membrane into the subdural space of cats fails